Evaluation of left ventricular structures in normotensive and hypertensive subjects by two-dimensional echocardiography: Anthropometric correlates in hypertension

Mr. Ugwu Anthony Chukwuka[*] MSc, Mr. Okwor Christian Anayo[†] BSc and Mr. Agbo Julius Amaechi[#] MSc

[*]Radiology Department, Federal Medical Centre, Ebonyi State, Nigeria
[†]Radiology Department, Ebonyi State University Teaching Hospital, Nigeria
[#]Department of Radiography, University of Nigeria, Enugu State, Nigeria

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ABSTRACT: This study was conducted with the objective to establish a nomogram for some left ventricular structures and their alterations in hypertension. Correlations between left ventricular structures and anthropometric variables in hypertension were also established. A sample of 320 normotensive and 80 hypertensive subjects were studied. Echocardiograhic end diastolic diameter, posterior wall thickness and septal wall thickness were obtained. Subject height, weight, age and blood pressures were obtained. Blood pressures were measured in sitting position. The values of left ventricular mass (LVM), left ventricular mass index (LVMI) and left relative wall thickness (RWT) were computed. Parametric tests were conducted. Tests were two tailed with $P < 0.05$ indicating statistical significance. Normal values of left ventricular structures were established; LVM: 63.72g – 336.18g, LVMI: 38.16g/m – 222.64g/m, and RWT: 0.25 – 0.52. Significant differences ($P < 0.05$) were established in LVM, LVMI and RWT between normotensive and hypertensive subjects. Positive and significant correlations were noted between these variables and systolic blood pressure in hypertensive subjects. A simple linear regression of RWT on Body surface area gives RWT = - 0.058 BSA + 0.475 in normotensive subjects. Normal values of left ventricular structures and a linear regression model have been established which could be used in the assessment of morbidity in hypertension.

KEYWORDS: Left ventricular mass; Left ventricular mass index; Relative wall thickness; Echocardiography; Hypertension

INTRODUCTION

Hypertension is defined as an elevation of the arterial blood pressure (BP). Its clinical significance is primarily due to morbid events affecting the heart and brain. Complications of hypertension, such as myocardial infarction and stroke, are not directly due to elevated pressures, but due to the resulting structural changes in the heart and blood vessels. Left Ventricular hypertrophy (LVH) is a structural consequence of hypertension and it is the strongest known predictor of cardiovascular morbidity and mortality[1,2].

(**Corresponding Author:** Mr. Ugwu Anthony Chukwuka, Imaging Scientist, Radiology Department, Federal Medical Centre, P.M.B 102, Ebonyi State, Nigeria, Email: tonybullng@yahoo.ca)

Echocardiographically determined LVH predicts these clinical outcomes both in individuals with hypertension[3] and healthy individuals[4], independent of other conventional risk factors. Increase in relative wall thickness (RWT), Left ventricular Mass (LVM) and left ventricular mass index (LVMI) have been reported in hypertension.[5] LVH is a consequence of increased ventricular afterload due to chronically elevated BP. Correlations between BP and LVM are relatively weak[6], which may reflect the inability of BP measurement to capture the dynamic nature of BP during daily life or to accurately reflect the afterload placed on the left ventricle over the course of a normal day. Ambulatory BP measured throughout the day is a better predictor of cardiac end-organ involvement than resting BP, suggesting the possibility that stress effects on blood pressure may be important.[7]

Left ventricular size has been assessed by an apical biplane modified Simpson's rule algorithm obtained by computer assisted planimetiry.[8] Echocardiographic measurements of the left ventricular dimensions and wall thicknesses at end diastole and end systole have been reported.[9] Nomograms of values of relative wall thickness, left ventricular Mass and left ventricular mass index, to the best of our knowledge do not exist for any African population. Internet search, using the available medical search engines did not reveal any report on the relative sensitivities of these cardiac parameters (RWT, LVM and LVMI) in the assessment of morbidity in hypertension. This study investigated the normal values of RWT, LVM and LVMI in this locality and also established their correlations with come anthropometric variables in hypertension.

MATERIAL AND METHOD

A total of 400 subjects of Ibo tribe were studied in Ebonyi State between June 2006 and April 2007. 320 normal subjects were recruited as volunteers (160 males and 160 females). Their ages ranged from 18 years to 72 years with Mean ages ± standard deviations of 40.59 ± 16.41 and 38.39 ± 11.35 for men and women respectively. A convenient and purposive sample of 80 hypertensive subjects was evaluated.

Exclusion criteria for both groups include sickle cell disease, obstructive airway disease, other cadiomyopathies, immune suppression, abdominal mass, pregnancy and diabetes. 22 of the hypertensive patients were women and 58 men. Their ages ranged from 27 years to 75 years. The mean ages ± standard deviations were 53.59 ± 10.66 and 48.64 ± 11.33 for men and women respectively. The hypertensive subjects were recruited from the out patient clinic. Subjects were classed as normotensive if their blood pressures were <140/90mmHg.[10] subjects gave their consent to participate in the study and institutional ethics approval was obtained.

Echocardiography studies were performed with SL I, siemens Medical System, USA Inc, Ultrasound Group, Issaquah WA with a 3.5 MHz sector in parasternal long and short axis views with the patient in either supine or left posterior oblique positions.[11] 2 D m-mode tracing was obtained in the short axis view. The following cardiac parameters were measured using m mode of 2 D derived image of the heart: posterior wall thickness (PWT) in diastole, septal wall thickness (SWT) in diastole and end diastolic diameter (EDD). Relative wall thickness was calculated as the ratio of 2 × (posterior wall thickness/end diastolic diameter).[5] Left ventricular mass was calculated according to the formula of Penn as left ventricular mass (g) = 1.04 [(end diastolic diameter + septal wall thickness + posterior wall thickness)^3 – (end diastolic diameter)^3] – 13.6g.[12] Left ventricular mass was indexed to height in grams per metre.[1] Measurements of left ventricular mass were taken just below the tip of the mitral valve.[5]

The body weights were measured on a bathroom scale Model H 89 LT Blue and height measured on a calibrated vertical wall. The body mass index (BMI) was calculated as Weight/Height^2, While body surface area (BSA) was calculated using the formula derived by Du Bois and Du Bois[13] thus: BSA = (Weight $^{0.425}$ × Height $^{0.725}$) × 0.007184.

The blood pressure (BP) was measured with a mercury sphygmomanometer in sitting position and was standardized for cuff size and position. SBP and DBP represent systolic and diastolic blood pressures.

Data Analysis: No sex categorization of data was done, as a previous study[14] reported no significant relationship between LVMI and gender. SPSS 11.0 software was used for statistical analysis. Summary statistics were generated. Inferential statistics were carried out using Pearson's correlation and unpaired t-tests. Tests were two – tailed with P <0.05 indicating statistical significance.

RESULTS

Table 1 Shows a descriptive statistics of all the computed cardiac parameters in hypertensive and normotensive subjects.

Table 2 Shows Pearson's correlation coefficients (r) and P values between all the computed cardiac parameters and anthropmetric variables in hypertensive subjects.

Unpaired t – test showed that there are significant differences in PWT, LVM and LVMI among normotensive and hypertensive subjects. The highest correlation coefficient between blood pressure and computed cardiac parameters occurred with RWT and SBP (r = 0.60, P = 0.00). A significant correlation was noted between BAS and RWT in normal subjects. A simple linear regression of RWT on BSA in normotensive subjects gave: RWT = - 0.058 BSA + 0.475.

Table1: Descriptive statistics of all the computed cardiac parameters in hypertensive and normotensive subjects

Group	LVM	LVMI	RWT
Hypertensive subjects			
Mean	317.06	196.92	.4472
Std. Error of Mean	6.70	4.455	.00941
Minimum	234.80	148.61	.34
Maximum	411.50	265.48	.64
Std. Deviation(SD)	42.37797	28.180	.05949
Kurtosis	-.237	.106	2.632
Skewness	269	.669	1.208
Normotensive subjects			
Mean	164.64	103.32	378
Std. Error of Mean	4.03	2.50	.0036
Minimum	63.72	38.16	.28
Maximum	336.18	222.64	.52
Std. Deviation(SD)	50.96	31.64	.0462
Kurtosis	.867	1.497	.042
Skewness	.706	.787	.298

Table 2: Pearson's correlation coefficients (r) and P values between all the anthropometric variables in hypertensive subjects

Cardiac parameters	Age (years)	Height (m)	Weight (kg)	SBP (mm Hg)	DBP (mm Hg)	BMI (kg/m^2)	BSA (m^2)
RWT	r = 0.11 P = 0.5 (NS)	r = - 0.07 P = 0.69 (NS)	r = - 0.134 P = 0.41 (NS)	r = 0.60 P = 0.00 (S)	r = 0.265 P = 0.127 (NS)	r = - 0.158 P = 0.33 (NS)	r = - 0.12 P = 0.46 (NS)
LVM	r = 0.058 P = 0.722 (NS)	r = 0.041 P = 0.80 (NS)	r = - 0.103 P = 0.53 (NS)	r = o.488 P = 0.001 (S)	r = 0.21 P = 0.20 (NS)	r = - 0.199 P = 0.219 (NS)	r = - 0.06 P = 0.72 (NS)
LVMI	r = 0.12 P = 048 (NS)	r = - 0.31 P = 0.05 (NS)	r = - 0.36 P = 0.02 (NS)	r = 0.42 P = 0.01 (S)	r = 0.114 P = 0.484 (NS)	r = - 0.293 P = 0.066 (NS)	r = - 0.363 P= 0.021 (S)

r = Correlation Coefficient, S = Significant, NS = Not Significant

DISCUSSION

An exaggerated rise in a patient's blood pressure seems to be a common phenomenon after an echocardiographic examination. This response to mild stress was found to be related to structural alterations of the heart including a significant increase in left ventricular mass and an increased prevalence of left ventricular hypertrophy.[5] Moreover, the extent of blood pressure rise is independently associated with left ventricular mass and left ventricular hypertrophy.

The present data has given a nomogram for LVM, RWT and LVMI in this population. The striking thing about these nomograms is the obvious overlap between normotensive and hypertensive subjects. Even though statistically significant differences exist between LVM, RWT, and LVMI in normotension and hypertension, the implication is that some hypertensive subjects would have normal values and vice versa. This implies that these nomograms derived in normotensive subjects may not be the best clinical baseline or benchmark in assessing morbidity in hypertension. Using prediction models derived from normal subjects to estimate normal values (± 2 Standard Deviation) may give better results. In normotensive subjects, RWT did not significantly correlate with BPS and BPD while LVM and LVMI correlated significantly with BPD (unpublished data, 2007). In the present study, RWT has the strongest correlation with blood pressure (BPS) in hypertensive subjects. This suggests that RWT is more sensitive to hypertension than LVM and LVMI. The authors hence suggest that RWT could be a more potent factor in the assessment of morbidity in hypertension. A linear regression equation of RWT on body surface area which correlated significantly with RWT (unpublished data, 2007) was derived thus: RWT (± 2 SD) = - 0.058 BSA + 0.475. This derivation was made on normal subjects, hence the 2 SD in normal subjects should be used to established a normal range of RWT. This study is consistent with a previous study[5], which reported increase in RWT, LVM and LVMI in hypertension. The result of this study partly agrees with the findings of a previous study[15] which reported no significant relationship between LVMI and age and also noted that LVMI significantly correlated with both weight and BMI. By contrast, this study reported no significant correlation between LVMI and BMI. LVMI also correlated with SBP in the previous study[15] and the present study.

In this study, SBP was the single most robust predictor of LVM, LVMI and RWT. This finding is not in agreement with a previous study[15], which found BMI as the single most robust predictor of LVMI. In this previous study, correlation and regression analyses were conducted using raw LVM. As would be expected, the overall magnitude of association for hemodynamic variables were substantially weaker with raw LVM compared with LVMI, but importantly the pattern of observations across the various BP measurement contexts remained fundamentally similar for raw LVM to those reported for LVMI. A similar thing was noticed in this study where the pattern of significance was the same across BP measurement (SBP and DBS) but the overall magnitude of association for other anthropometric variables were more and significantly in favor of LVMI.

To the best of our knowledge, this is the first time a prediction model for the estimation of RWT is being established in this locality. One obvious limitation of this study was that the two groups were not BMI and age matched. Future studies in this locality and elsewhere should take care of this limitation. Longitudinal studies are necessary to assess the possible effects of anthropometric, morphometric and environmental factors on the development of left ventricular hypertrophy. Replication of this study in other ethnic and racial nationalities is also suggested.

Finally, this study has established a nomogram for LVM, LVMI, RWT and a prediction equation for RWT both of which would be useful in the assessment of morbidity in hypertension.

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